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EXAMINER	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/586,288	DOGULU ET AL.	
	Examiner	Art Unit	
	AMANDA SHAW	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 June 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,8,10-25 and 29* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,8,10-25 and 29* is/are rejected.

7)☒ Claim(s) *23-25 and 29* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *6/19/2008*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. This action is in response to the amendment filed June 19, 2008. This action is made FINAL.

Claims 1, 8, 10-25 and 29 are currently pending. Claims 1, 8, 10-13, 18-25, and 29 have been amended.

Withdrawn Rejections

2. The rejections made under 35 USC 112 2nd paragraph in sections 8-10 of the Office Action of March 17, 2008 are withdrawn in view of amendments made to the claims.

Elections/Restrictions

3. The amended claims are still not commensurate in scope with the election of all of the 143 mutations or polymorphisms in Table 1. Specifically the Applicants were required to elect a single invention, i.e., “a single SNP in each of the 8 genes or a single combination of SNPs in each of the 8 genes”. In response to this requirement the Applicants elected "the 143 mutations or polymorphisms listed in Table 1". In the instant case the claims require "screening the human subject for the presence of the 143 mutation or polymorphisms listed in Table 1, wherein the presence of one or more of the mutations or polymorphisms indicates that the human subject has a genetic predisposition”. The “one or more” language is problematic because a search of the claims as presently written would require at least 143 separate searches and

consideration of any prior art relevant to each mutation or polymorphism searched. As such this places a serious burden on the examiner and on the Office. Again it is reiterated that the Applicants elected in the response filed November 16, 2007 the combination of all of the 143 mutations or polymorphisms listed in Table 1. Therefore claim 1 must be amended to recite "wherein the presence of the 143 mutations or polymorphism indicates that the human subject has a genetic predisposition for VT". Additionally claims 11-13, 22-23 and 29 must be amended to remove reference to " one or more of the mutations or polymorphisms", "one or more of the mutated sequences" and "at least one mutation or polymorphism".

For examination purposes the claims have been examined to the extent that the read on the elected invention which requires "screening the human subject for the presence of the 143 mutation or polymorphisms listed in Table 1, wherein the presence of all of the mutations or polymorphisms indicates that the human subject has a genetic predisposition".

Claim Objections

4. Claims 23-25 and 29 are objected to because they does not properly depend from claim 1 (in the case of claims 23-25) and claims 1, 10-13 (in the case of claim 29). As stated in MPEP 608.01(n), "The test as to whether a claim is a proper dependent claim is that it shall include every limitation of the claim from which it depends (35 U.S.C. 112, fourth paragraph) or in other words that it shall not conceivably be infringed by anything which would not also infringe the basic claim. On the other hand, if claim 1

recites a method of making a specified product, a claim to the product set forth in claim 1 would not be a proper dependent claim since it is conceivable that the product claim can be infringed without infringing the base method claim if the product can be made by a method other than that recited in the base method claim." Specifically Claims 23-25 are objected to because they do not properly depend from claim 1. In the present situation, claims 23-25 are drawn to a method of selecting a venous thrombosis therapy yet these claims depend from claim 1 which is drawn to a method of detecting genetic predisposition to VT. Further Claim 29 is objected to because it does not properly depend from claim 13. In the present situation, the method of claim 29 is drawn to a method of detecting a genetic predisposition to venous thrombosis in a human subject. This claim requires the step of applying the amplification products of claim 13 to an array. Claim 13 depends from claims 1, 10, 11, and 12.

Claim Rejections - 35 USC § 112

5. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 10-18 and 21 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 10 and 18 recite the limitation "the VT related molecules". There is insufficient antecedent basis for this limitation in the claim because the claims do not previously refer to “VT related molecules”.

Claim Rejections - 35 USC § 112

6. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1, 8, 10-25 and 29 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The test of enablement is whether one skilled in the art could make and use the claimed invention from the disclosures in the application coupled with information known in the art without undue experimentation (*United States v. Teletronics Inc,* 8 USPQ2d 1217 (Fed Cir. 1988)). Whether undue experimentation is needed is not based on a single factor, but rather a conclusion reached by weighing many factors (See *Ex parte Forman*, 230 USPQ 546 (Bd. Pat. App. & Inter. 1986)) and *In re Wands* 8 USPQ2d 1400 (Fed. Cir. 1988)).

The breadth of the claims and nature of the invention

Claims 1, 8, 10-22 and 29 are drawn to a method of detecting genetic predisposition to venous thrombosis (VT) in a human subject, comprising: screening the human subject for the presence of the 143 mutations or polymorphisms listed in Table 1 wherein the presence of the 143 mutations or polymorphisms indicates that the human subject has a genetic predisposition for VT. Thus the claims encompass a method wherein the human individual must have **all** of the 143 recited mutations or polymorphisms in order to have a genetic predisposition to VT. Claim 8 further requires that the method provide a probability of developing VT of at least 98% in Caucasians, at least 85% in Asians, and at least 87% in Africans.

Claim 22 is drawn to a method of detecting genetic predisposition to VT in a human subject, comprising: applying amplification products to an array, wherein the array comprises oligonucleotide probes capable of detecting the 143 mutations or polymorphisms listed in Table 1, and analyzing the hybridization complexes to determine if the amplification products comprise the 143 mutations or polymorphisms listed in Table 1 wherein the presence of the 143 mutations or polymorphisms indicates that the human subject has a genetic predisposition for VT. Thus the claims encompass a method wherein the human individual must have **all** of the 143 recited mutations or polymorphisms in order to have a genetic predisposition to VT.

Claim 29 is drawn to a method of detecting a genetic predisposition to venous thrombosis (VT) in a subject, comprising: applying amplification products to the array of claim 13, wherein the amplification products comprise amplified nucleic acids obtained from the subject, wherein the nucleic acids comprise coding or non-coding sequences

from AT III, protein C, protein S, fibrinogen, factor V, factor II, MTHFR **and** ACE. incubating the amplification products with the array for a time sufficient to allow hybridization between the amplification products and oligonucleotide probes, thereby forming amplification products: oligonucleotide probe complexes; and analyzing the amplification products: oligonucleotide probe complexes to determine if the amplification products comprise **all** of the 143 mutations or polymorphisms in the AT III, protein C, protein S, fibrinogen, factor V, factor II, MTHFR, or ACE genes, wherein the presence of **all** of the 143 mutations or polymorphisms indicates that the subject has a genetic predisposition for VT.

Since the claims do not specify the ethnicity of the patients examined, the claims broadly encompass detecting predisposition to VT in human subjects of any ethnicity, including Caucasians, African Americans, Koreans, Chinese, etc. Also, claim 8 requires probability of developing VT of at least 98% in Caucasians, at least 85% in Asians, and at least 87% in Africans, however, independent claim 1 does not provide any steps for determining a probability and therefore it is unpredictable to what probability the claim is referring and therefore also unpredictable to achieve said probabilities by the claimed method.

Guidance in the Specification and Working Examples

The specification characterizes 143 mutations and polymorphisms as being associated with venous thrombosis (pp 70-71, table 1). However, in example 1, the specification only teaches analyzing 10 of the VT associated susceptibility alleles for calculations of logistic regression and likelihood ratios for predicting the probability of

developing VT, and the specification teaches that the data used for the calculations were derived from previously reported studies (pg 71, lines 5-18 to pg 73). In Table 3 (pg 75), the specification teaches the likelihood ratios and probabilities for developing VT for each polymorphism assesses. However, the table does not teach which Antithrombin III polymorphism was analyzed to yield the data in the table. In addition, many of the likelihood ratios were only calculated for certain ethnic populations: for example, the fibrinogen Thr312A1a polymorphism was only analyzed in Caucasian populations. Therefore, it is unclear from the data in the specification how to use each of the 10 analyzed polymorphisms/mutations to determine genetic predisposition to venous thrombosis in any population, and thereby is unpredictable to use any of those polymorphisms/mutations to determine genetic predisposition to venous thrombosis in any population. Further, since there is no data in the specification for the remaining 133 polymorphisms it is unpredictable to use any of the 133 polymorphisms/mutations to determine genetic predisposition to venous thrombosis in any population.

Finally, the specification states that concurrent screening of the 8 genes results in a 99.7% probability of developing VT in Caucasian populations, an 85.1% probability in Asian populations and an 88.7% probability in African populations (pp 75-76). However, it is unclear how these numbers were calculated for each population, since only 3 of the 8 polymorphisms/mutations were studied in all 3 populations. Further, the specification states that, e.g. the FV Cambridge G to C mutation at position 1091 has only been described in Caucasian populations (pg 51). Therefore, based on the lack of descriptive data in the specification, along with statements that certain mutations are

only known in Caucasian populations, it is unpredictable to use the combination of 8 polymorphisms/mutations to predict genetic predisposition to venous thrombosis in any ethnic population.

The unpredictability of the art, the state of the prior art, and the level of skill in the art

While the state of the art and level of skill in the art with regard to correlating polymorphisms or mutations with disease state is high, the level of unpredictability in associating any polymorphism or mutation with a particular disease state is even higher. The level of unpredictability is demonstrated by the prior art, the post filing art, and the instant specification.

Nagaraja (Nagaraja, et al. Journal of Clinical Neuroscience, July 2007; 14(7):635-638) teaches studying the prevalence of the prothrombin G20210A variant in south Indian women and examine its association with cerebral veno-sinus thrombosis (CVT)—a type of venous thrombosis— occurring during puerperium in these women. Nagaraja teaches that studies evaluating the prothrombin variant in CVT report frequencies varying from 0 to 50% and that differences in the frequencies reported in those studies might be explained by ethnic differences (pg 637, col 1). Nagaraja also teaches an absence of the G20210A variant in women their study and suggest that the result is due to absence of the G20210A variant in the general Indian population (pg 637, col 1). Therefore, based on the data described by Nagaraja, it is unpredictable to use the prothrombin G20210A variant to predict predisposition to VT in any Asian population, and further, based on the summary of previous studies by Nagaraja it is also

unpredictable to use the G20210A variant to predict predisposition to VT in any human population.

Bezemer (Bezemer, et al. Arch Intern Med. 2007 Mar 12;167(5):497-501). Bezemer teaches a single large study on the association between MTHFR 677C→T polymorphism and venous thrombosis that included 4375 patients with a first venous thrombotic event, either deep vein thrombosis of the leg or pulmonary embolism, and 4856 control subjects from the Multiple Environmental and Genetic Assessment of risk factors for venous thrombosis (MEGA Study) (pg 498, col 1). Bezemer teaches that the patients were all Dutch speakers and that the study was performed in the Netherlands. Bezemer teaches that no evidence was found for an association between MTHFR 677C→T and the risk of venous thrombosis. Therefore, based on the results of this study, it is unpredictable to use the MTHFR 677C→T polymorphism to predict genetic predisposition to venous thrombosis in Dutch patients and thereby in any population.

Halushka (Halushka, et al. Nature Genetics, 1999; 22:239-247) teaches assessing the age or ancestral state of human SNP alleles by obtaining the corresponding orthologous sequence from the closely related great apes (page 244, column 1, paragraph 2). Halushka teaches that although there was a high degree of sequence identity between great ape and human samples (page 244, column 2, paragraph 1), the average nucleotide divergence between human and chimpanzee, gorilla and orangutan was 0.010, 0.012 and 0.021, respectively, and that and that these values are more than ten times greater than the within-population human diversity (page 244, column 2, paragraph 1). Halushka teaches that the data suggest that 95% of

population-specific SNPs arose in the human lineage after population differentiation and that the common allele [between human and ape] is the likely ancestral state.

Regarding using polymorphisms to make predictions, the art teaches genetic variations and associations are often irreproducible. Hirschhorn (Hirschhorn et al. Genetics in Medicine. Vol. 4, No. 2, pages 45-61, March 2002) teaches that most reported associations are not robust. Of the 166 associations studied three or more times, only 6 have been consistently replicated. Hirschhorn suggests a number of reasons for the irreproducibility of studies, suggesting population stratification, linkage disequilibrium, gene-gene or gene-environment interactions, and weak genetic effects and lack of power are possible factors that lead to such irreproducibility. Hirschhorn et al. caution that the current irreproducibility of most association studies should raise a cautionary alarm when considering their use as diagnostics and prognostics (p. 60, Col. 2). Thus, Hirschhorn cautions in drawing conclusions from a single report of an association between a genetic variant and disease susceptibility.

Ioannidis (Ioannidis. Nature Genetics, Vol. 29, pages 306-309, November 2001) teaches that the results of the first study correlate only modestly with subsequent research on the same association (abstract). Ioannidis teaches that both bias and genuine population diversity might explain why early association studies tend to overestimate the disease protection or predisposition conferred by a genetic polymorphism (abstract). Therefore, it is unpredictable to use genetic associations for diagnostic purposes.

Kroese et al. (Kroese et al., Genetics in Medicine, vol 6 (2004), p. 475-480) teach

genetic tests are heterogeneous in nature and the exact characteristics of a particular genetic test to be evaluated must be tightly defined. Kroese et al. teach that a particular genetic condition may be caused by more than one gene and these variations may be due to deletions and insertions not detected by routine sequence methods (see page 476, 2nd column, last paragraph). Kroese et al. teach that genetic test is shorthand to describe a test to detect a particular genetic variant for a particular disease in a particular population and for a particular purpose and that it should not be assumed that once the characteristics of a genetic test are evaluated for one of these reasons that the evaluation will hold or be useful for other purposes and all measures of the test performance should be presented with their 95% confidence intervals (see page 477, 1st column, 1st and 2nd full paragraph). Kroese et al. teach that the limitations of our genetic knowledge and technical abilities means that for the moment there are likely to be gaps in the information needed to complete a thorough evaluation of many genetic tests (see page 479, 2nd column, last paragraph).

Quantity of Experimentation

The claims are drawn to a method of detecting genetic predisposition to venous thrombosis (VT) in a human subject, comprising: determining whether the any subject has the 143 mutations or polymorphisms listed in Table 1, wherein the presence of the 143 mutations or polymorphisms indicates that the subject has a genetic predisposition for venous thrombosis. The specification teaches studying 10 of the 143 polymorphisms and Table 3 indicates that 7 of the polymorphisms were not studied in all ethnic populations. Also, while the specification indicates in Table 1 that, for

example, a Caucasian patient with the MTHFR 677C→T SNP has a 0.16% probability of developing VT, Bezemer teaches in a study of Dutch patients that there is no association between the MTHFR 677C→T SNP and VT. Since many Dutch people are also Caucasian, the data from Bezemer and in the specification conflict with regard to the use of MTHFR C677T as predictive of VT, and therefore it is unpredictable to use MTHFR C677T as a means of predicting genetic predisposition to VT in Caucasian populations. Also, while the specification teaches that the Prothrombin G20210A SNP when present in a Caucasian patient indicates a 0.2% probability of developing VT, Nagaraja teaches that studies evaluating the prothrombin variant in CVT report frequencies varying from 0 to 50% and Nagaraja teaches an absence of the G20210A variant in women their study and suggests that the result is due to absence of the G20210A variant in the general Indian population. Therefore, due to conflicting data from the combination of results from Nagaraja, other reported studies, and the data in the specification, it is unpredictable to use of the G20210A variant to predict genetic predisposition to VT. Finally, Hirschhorn, Ioannidis and Kroese teach that the association between SNPs and disease is often unpredictable due to the number of variable associated with performing such studies, variables including population diversity, linkage disequilibrium and gene-environment interactions. Therefore, the skilled artisan would be required to perform a very large study to determine if the combination of 143 polymorphisms can be used in any ethnic population to predict genetic predisposition to VT. This would require undue and unpredictable experimentation with no expectation of success.

Claim 8 requires that the method of claim 1 provide a probability of developing VT of at least 98% in Caucasians, at least 85% in Asians, and at least 87% in Africans. The specification teaches that concurrent screening of the 8 genes results in a probability of VT of 99.7% in Caucasian, an 85.1% probability in Asians and an 88.7% probability in Africans. However, the data in the table also indicates that only 3 of the 10 SNPs were studied in the three ethnic populations. Therefore it is unclear how the values were calculated for all 8 genes, and since there is no data for the other 133 mutations, it is unclear what the values would be for the combination. In addition, the method does not provide a means of determining the claimed probabilities. Therefore, it is unpredictable to use the combination of 143 SNPs to determine a probability of developing VT of at least 98% in Caucasians, at least 85% in Asians, and at least 87% in Africans, and the skilled artisan would be required to perform a large study to identify a formula to use that would result in all 143 of the SNPs/mutations yielding a probability of developing VT of at least 98% in Caucasians, at least 85% in Asians, and at least 87% in Africans. This would require undue and unpredictable experimentation with no expectation of success.

Conclusion

Given the lack of data from all organisms, the conflicts between the art and specification on polymorphisms that are associated with VT, and the lack of guidance in the claims and specification for how to perform the claimed methods, methods of detecting genetic predisposition to venous thrombosis (VT) are replete with unpredictable experimentation that is considered undue.

Thus given the broad claims in an art whose nature is unpredictable, the unpredictability of that art, the large quantity of research required to define these unpredictable variables, the lack of guidance provided in the specification, and the negative teachings in the prior art balanced only against the high skill level in the art, it is the position of the examiner that it would require undue experimentation for one of skill in the art to perform the methods of the claims as broadly written.

Response To Arguments

7. In the response filed June 19, 2008, Applicants traversed the enablement rejection.

Specifically the Applicants argue that what Table 3 shows is that any Antithrombin III polymorphism associated with VT is found there is a 1.6% probability of developing VT. The further argue that it is irrelevant that the probability data for each polymorphisms is not shown. The Applicants assert that Table 3 clearly shows that instead of analyzing individual genes, polymorphism, mutations, or populations, the use of all eight genes permits one to determine a subject's genetic predisposition to developing VT with a much higher probability (e.g., 1.6% vs 85.1% to 99.7%. This probability is at least 98% in Caucasians, at least 85% in Asians, and at least 87% in Africans.

This argument has been fully considered but is not persuasive. In the instant case the claims require screening for the presence of the 143 mutations or polymorphisms listed in Table 1, wherein the presence all of the 143 mutations or

polymorphisms indicates that the human subject has a genetic predisposition for VT. Thus the nature of the claimed invention requires the knowledge of a reliable association between each of the 143 mutations and VT. Yet the Applicants have not provided any evidence that each of the 143 mutations on their own or in combination were found to be associated with VT. The first problem with the data presented in the specification is not clear how the likelihood ratios and posterior probabilities were calculated. For example it's not clear how the Applicants arrived at 349250.7 for the likelihood ratio of concurrent screening of 8 genes. Further it is unclear if this number is based on a person who had all of the 10 mutations or only at least one of the 10 mutations. Further it is unclear how this number translated into a posterior probability of 99.7%. While data presented in Table 3 may show concurrent screening of 10 non disclosed mutations in 8 genes is superior to screening each mutation on its own, it is highly unpredictable whether the concurrent screening of all of the 143 mutations (derived from 8 genes) listed in Table 1 will also be superior to screening each of the 143 mutations on its own. This is due to the fact that some mutations will have a stronger association with VT disease than others. Further it is highly unlikely whether there even exists a human individual that has all 143 mutations. For these reasons the enablement rejection is maintained.

Conclusion

8. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP

§ 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Amanda M. Shaw whose telephone number is (571) 272-8668. The examiner can normally be reached on Mon-Fri 7:30 TO 4:30. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached at 571-272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Amanda M. Shaw
Examiner
Art Unit 1634

/Carla Myers/
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